

Right atrial laceration *Complication of external cardiac massage*

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Right atrial laceration is described as a complication of external cardiac massage in two patients. In each there was a pre-existent cause of weakness of the atrial wall. There was pronounced right atrial dilatation with attenuation of the wall in one case, and in the other there was a granulomatous myocarditis involving the atrial wall. The significance of atrial laceration as a complication of traumatic injuries to the heart is discussed.

Stephenson (1969) has comprehensively reviewed traumatic lesions of the heart after resuscitative efforts for cardiac arrest. In only one case, however, has laceration of the right atrium been recorded as a result of external cardiac massage (Wolfe, Dudley, and Wallace, 1968).

Recently within a period of one week we have had the opportunity of examining two patients *post mortem* who had received terminal external cardiac massage and in whom lacerations of the right atrium were found. In view of the rarity of this complication of cardiac resuscitation and of the discovery of an underlying condition in each case which could have predisposed to rupture, we feel that the findings should be reported.

Case reports

Case 1

A man, aged 60, with a 10-year history of symptoms relating to rheumatic heart disease, was admitted to hospital in gross heart failure and with electrocardiographic evidence of digitalis toxicity (bigeminal rhythm). The patient collapsed shortly after admission and resuscitative efforts including external cardiac massage were unsuccessful.

The following were the relevant post-mortem findings.

Thorax There was a transverse fracture of the upper sternum, 2 cm below the manubrio-sternal junction and bilateral fractures of all ribs anteriorly 2 to 3 cm lateral to the costochondral junctions.

Pericardial sac This contained 1000 ml of fresh blood.

Heart (540 g) The right atrium was considerably dilated, thinwalled, and showed a transverse laceration (4 × 1.5 cm) through all coats of the posterolateral wall

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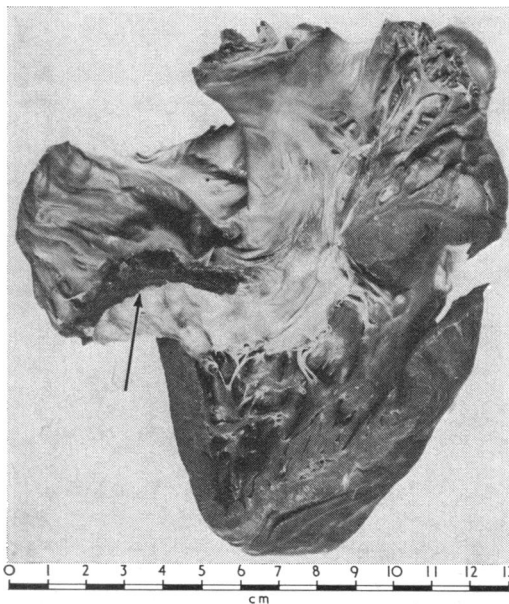


FIG. 1 Case 1. Transverse laceration of right atrium (arrowed).

(Fig. 1). This laceration was situated 7 mm above the tricuspid valve. The latter was incompetent with mild fibrous thickening and adhesion of its cusps. The chordae tendineae were shortened and thickened. The right ventricle was hypertrophied (8 mm) and slightly dilated. The left atrium was much dilated with an attenuated wall (2 mm). The endocardium was rough, opaque, and partly covered by a thin layer of mural thrombus. The mitral valve showed 'button-hole' stenosis (2.5 × 0.2 cm) and shortening and thickening of the chordae tendineae. The left ventricle was not hypertrophied

(13 mm) or dilated. The aortic valve was stenosed, with fusion of its heavily calcified cusps. The histological findings confirmed a healed rheumatic carditis with valvulitis.

Case 2

An 83-year-old woman, on admission to hospital, gave a 3-week history of severe breathlessness with increasing incapacity. On examination she was in atrial fibrillation and congestive heart failure. Three days after admission she collapsed suddenly and resuscitative measures involving external cardiac massage and intracardiac injections were unsuccessful. Relevant findings at necropsy were as follows.

Thorax There was a transverse fracture of the sternum 3 cm below the manubrio-sternal junction, with bilateral fractures of all ribs anteriorly just outside the costochondral junctions.

Pericardial sac This contained 2 to 3 ml of fresh blood.

Heart (420 g) Several needle puncture marks were present in the anterior surface of the left ventricle, but not in the right atrium. The right atrium was slightly dilated. There was a triangular laceration ($2 \times 2 \times 2$ cm) extending from the inferior margin of the fossa ovalis to involve the crista terminalis (Fig. 2). This laceration involved endocardium and myocardium but spared the epicardium.

The tricuspid valve was intact. The right ventricle was slightly dilated and myocardial thickness was 3 mm. The muscle was mottled in appearance with multiple small pale areas visible through the endocardium (Fig. 3). The left atrium was normal. The left ventricular wall was slightly hypertrophied. The muscle was soft and showed slight mottling and streaking on the cut surfaces (Fig. 4).

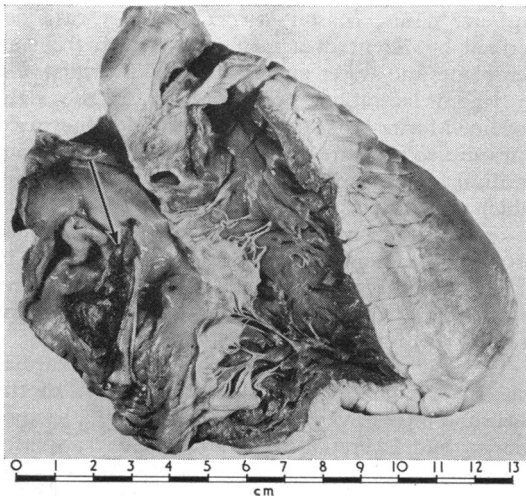


FIG. 2 Case 2. Triangular laceration of right atrial wall (arrowed).

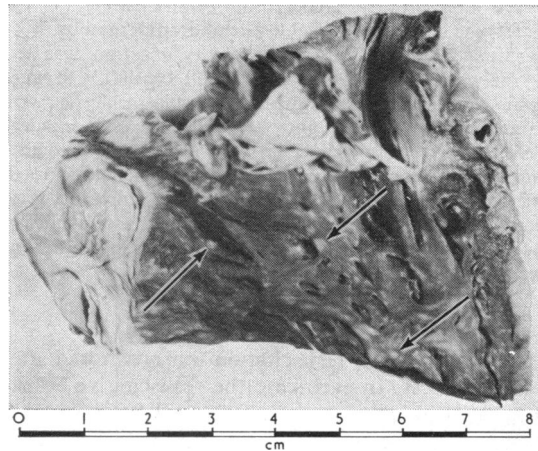


FIG. 3 Case 2. Several small pale inflammatory foci (arrowed).

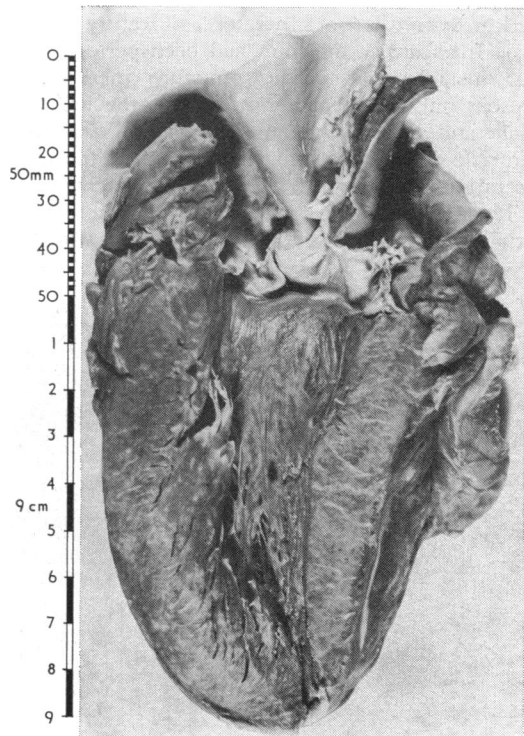


FIG. 4 Case 2. Mottling of cut surfaces of left ventricular wall.

Histologically the myocardium of all chambers of the heart was involved by a widespread **granulomatous myocarditis** (Fig. 5). The inflammatory reaction was extremely intense and of a mixed cell type. There were some neutrophil polymorphs and plasma cells with larger numbers of lymphocytes and large mononuclear cells. Many eosinophil cells were noted and moderate numbers of giant cells of the Langhans type had formed. The small intracardiac coronary arteries showed a focal acute arteritis. In several areas there were small foci of necrosis in the muscle. Similar **granulomatous lesions** were noted in the liver.

Discussion

In these two cases resuscitation was attempted after cardiac arrest. In each case the resuscitative efforts resulted in high transverse fracture of the sternum with bilateral fractures of the ribs anteriorly producing a flail chest. The striking feature was the presence of right atrial lacerations. In the first case the laceration was transverse and situated on the posterolateral wall. The laceration involved the full thickness of the atrial wall causing a massive haemopericardium. In the second case the laceration was situated on the inferior margin of the fossa ovalis and involved the endocardium and myocardium but spared the epicardium. Massive haemopericardium was not, therefore, a feature of this case. Intracardiac injection had been performed in this instance but needle puncture marks were present only in the anterior wall of the left ventricle and not in the anterior wall of the right ventricle or right atrium. In both cases the atrial lacerations were remote from bone fragments.

The one previous published case where right atrial laceration occurred in association with ex-

ternal cardiac massage was that described by Wolfe and his co-workers (1968). The patient was a 69-year-old lady admitted for evaluation and treatment of severe and progressive congestive heart failure. At necropsy the patient had a flail chest with bilateral haemothorax. There was also a haemopericardium resulting from a 4.5 cm full thickness supraventricular laceration of the anterior wall of the right atrium.

Our two cases differed in that the lacerations were not present on the anterior wall of the atrium and damage caused by bone fragments could be excluded with certainty. Extensive fractures of the rib cage were found in both patients and these findings might indicate that the force applied during external cardiac massage may have, in this instance, been somewhat excessive, though the presence of osteoporosis in both cases was probably a more important factor. Wolfe did not specifically exclude this possibility in his case. Also in our cases there were factors causing pre-existing weakness to the atrial wall. In the first case the atrium was dilated with conspicuous attenuation of the muscle because of tricuspid regurgitation. In the second case the myocardium was involved by a widespread **granulomatous myocarditis** associated with small focal areas of necrosis. These lesions were similar to those in a case described by Waugh (1952) in which there was strong circumstantial evidence of an association with penicillin hypersensitivity. However, evidence of such an association could not be elicited in the present case.

It seems most likely that the atrial lesions were caused by a sudden abrupt rise in intracardiac pressure caused by external cardiac massage.

This concept derives support from several authors. Thus, Mason (1973) quotes a case described by Krefft of a man who ejected from an aeroplane into water and died from drowning and in whom lacerations were present in the right atrium. Moritz (1954) stated that sudden increased intracardiac pressure after an impact over the praecordium was an important cause of cardiac rupture which may be partial or complete and may be near to or remote from the site of impact. Moritz also listed the common sites of traumatic rupture in order of diminishing frequency as: right atrium, left ventricle, right ventricle, left atrium, interventricular septum, and valves.

The traumatic complications of external cardiac massage previously reported include tears of the aorta and inferior vena cava, pneumothorax, haemothorax, and laceration of the lungs, liver, spleen, stomach, or colon (Stephenson, 1969). Many of the extracardiac complications mentioned above are at least potentially amenable to treatment if resuscitation is successful. On the other hand with traumatic

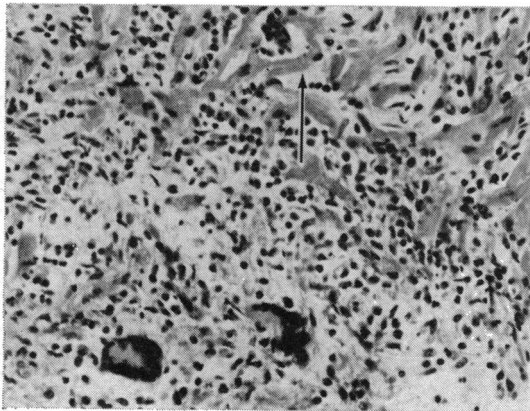


FIG. 5 Case 2. Giant cell granulomatous myocarditis. Separated myocardial fibres (arrowed). ($\times 175$.)

cardiac lesions in which the development of haemopericardium is a particular hazard resuscitative efforts are doomed to failure. In these circumstances a fatal outcome is inevitable.

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